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EXAMINER	
LEE, G	
ART UNIT	**PAPER NUMBER**
1632	7

DATE MAILED: 09/27/00

Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary

Application No.	Applicant(s)
09/357,675	Croce, Carlo M.
Examiner	**Group Art Unit**
Gai (Jennifer) Mi L e	1632

☐ Responsive to communication(s) filed on _____

☐ This action is **FINAL.**

☐ Since this application is in condition for allowance except for formal matters, **prosecution as to the merits is closed** in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

A shortened statutory period for response to this action is set to expire 3 month(s), or thirty days, whichever is longer, from the mailing date of this communication. Failure to respond within the period for response will cause the application to become abandoned. (35 U.S.C. § 133). Extensions of time may be obtained under the provisions of 37 CFR 1.136(a).

Disposition of Claim

☒ Claim(s) *1-3, 10, 11, and 13-16* is/are pending in the applicat

Of the above, claim(s) *4-9 and 12* is/are withdrawn from consideration

☐ Claim(s) _____ is/are allowed.

☒ Claim(s) *1-3, 10, 11, and 13-16* is/are rejected.

☐ Claim(s) _____ is/are objected to.

☐ Claims _____ are subject to restriction or election requirement.

Application Papers

☒ See the attached Notice of Draftsperson's Patent Drawing Review, PTO-948.

☐ The drawing(s) filed on _____ is/are objected to by the Examiner.

☐ The proposed drawing correction, filed on _____ is ☐ approved ☐ disapproved.

☐ The specification is objected to by the Examiner.

☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).

☐ All ☐ Some* ☒ None of the CERTIFIED copies of the priority documents have been

☐ received.

☐ received in Application No. (Series Code/Serial Number) _____.

☐ received in this national stage application from the International Bureau (PCT Rule 17.2(a)).

*Certified copies not received: _____

☒ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

☒ Notice of References Cited, PTO-892

☐ Information Disclosure Statement(s), PTO-1449, Paper No(s). _____

☐ Interview Summary, PTO-413

☒ Notice of Draftsperson's Patent Drawing Review, PTO-948

☐ Notice of Informal Patent Application, PTO-152

--- SEE OFFICE ACTION ON THE FOLLOWING PAGES ---

DETAILED ACTION

Applicant's election of Group I, Claims 1-3 and 10-16 without traverse in Paper No. 6 is acknowledged.

Please note that it appears that claim 12, directed to a pharmaceutical composition comprising a therapeutically effective amount of the Nit1 protein, had been inadvertently and improperly placed in the Invention of Group I rather than in the Invention of Group II. In light of the compact prosecution, claim 12 has been properly rejoined with the Invention of Group II (claims 4-6 and 13-14), drawn to a protein and uses thereof, which would properly encompass a pharmaceutical composition comprising a therapeutically effective amount of the Nit1 protein.

Claims 4-9 and 12 are withdrawn from further consideration by the examiner, 37 CFR 1.142(b), as being drawn to a non-elected Inventions. It is noted that in the Response section of the Restriction, Applicants indicate that claims 4-9 have been canceled. However, cancellation of a claim must take place In the Claims section of the Response to a restriction requirement. As such, claims 4-9 has not been canceled.

Claims 1-3, 10-11 and 13-16 are under examination only in so far as the claimed invention is drawn to the elected invention of a nucleic acid and methods of gene therapy using such.

Claims 1-3, 10-11 and 13-16 are currently under examination.

Priority

Applicant has not complied with one or more conditions for receiving the benefit of an earlier filing date under 35 U.S.C.119 (e) as follows:

An application in which the benefits of an earlier application are desired must contain a specific reference to the prior application(s) in the first sentence of the specification (37 CFR 1.78).

Claim Objections

Claim 11 is objected to because of the following informalities: On line 2, a space should be inserted between Nit1 protein. Appropriate correction is required.

Claims 13- 15 are objected to because of the following informalities: The claims have not been amended to read on only the elected invention. Appropriate correction is required.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Written Description

Claims 1-3, 10-11 and 13-16 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. Applicant is referred to the interim guidelines on written description published December 21, 1999 in the Federal Register at Volume 64, Number 244, pp. 71440-71442 (also available at www.uspto.gov).

Vas-Cath Inc. v. Mahurkar, 19USPQ2d 1111 (Fed. Cir. 1991), clearly states that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession *of the invention.* The invention is, for purposes of the 'written description' inquiry, *whatever is now claimed.*" Vas-Cath Inc. v. Mahurkar, 19USPQ2d at 1117. The specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed."Vas-Cath Inc. v. Mahurkar, 19USPQ2d at 1116.

The instant specification does not contain a written description of the invention in such full, clear, concise, and exact terms or in sufficient detail that one skilled in the art can reasonably conclude that applicant had possession of the claimed invention at the time of filing.

The claims are drawn to a purified *NIT1* gene. In particular, wherein the gene is a human gene or mouse gene. The claims are also directed to an isolated nucleic acid of less than 100 kb comprising a nucleotide sequence encoding a Nit1 protein wherein the Nit1 protein is a human Nit1 protein (claims 10-11). The claims are further drawn to a method of treating or preventing a disease or disorder in a subject comprising administering to said subject a therapeutically effective amount of a molecule that inhibits or enhances Nit1 protein function (claims 13-14) or a method of gene therapy for treating or preventing a disease or disorder in a subject by using a vector containing the *NIT1* gene coding sequence (claims 16). Claim 15 is drawn to a method of diagnosing or screening for the presence of or a predisposition for developing a disease or disorder in a subject comprising detecting one or more mutations in *NIT1* DNA or RNA derived

from the subject in which the presence of said one or more mutation indicates the presence of the disease or disorder or a predisposition for developing the disease or disorder.

The specification discloses an isolated cDNA sequence, SEQ ID NO: 1, which encodes a predictive polypeptide sequence. Absent evidence to the contrary, the *NIT1* gene elected for examination is deemed to be an incomplete cDNA. Because the cDNAs that correspond to the SEQ ID NO: 1 mentioned in the specification are not full-length, a sequence prepared from undefined parts of a cDNA clone will not comprise the entire coding region of any particular gene, nor is it clear the partial sequence is even in frame to encode a polypeptide. The claims, as written, however, encompass polynucleotides which vary substantially in length and also in nucleotide composition. The broadly claimed genus additionally, encompasses *NIT1* genes, as well as genes incorporating only portions of the disclosed sequence.

The instant disclosure of a single species of nucleic acid does not adequately describe the scope of the claimed genus, which encompasses a substantial variety of subgenera including full-length genes. A description of a genus of cDNAs may be achieved by means of a recitation of a representative number of cDNAs, defined by nucleotide sequence, falling within the scope of the genus or of a recitation of structural features common to members of the genus, which features constitute a substantial portion of the genus. *Regents of the University of California v. Eli Lilly & Co.*, 119 F3d 1559, 1569, 43 USPQ2d 1398, 1406 (Fed. Cir. 1997). The instant specification fails to provide sufficient descriptive information, such as definitive structural or functional features of the claimed genus of polynucleotides. There is no description of the conserved regions which

are critical to the structure and function of the genus claimed. The specification proposes to discover other members of the genus by using a comparison with nitrilase and Fhit homologs which are encoded as fusion proteins in *D. melanogaster* and *C. elegans*. There is no description, however, of the sites at which variability may be tolerated and there is no information regarding the relation of structure to function. Structural features that could distinguish the compounds in the genus from others excluded are missing from the disclosure. Furthermore, the prior art does not provide compensatory structural or correlative teachings sufficient to enable one of skill to isolate and identify the polynucleotides encompassed and no identifying characteristic or property of the instant polynucleotides is provided such that one of skill would be able to predictably identify the encompassed molecules as being identical to those instantly claimed.

The specification further fails to identify and describe the 5' and 3' regulatory regions and untranslated regions essential to the function of the claimed invention, which are required since the claimed invention currently encompasses the gene. The art indicates that the structures of genes with naturally occurring regulatory elements and untranslated regions is empirically determined (Harris et al. J. of The Am Society of Nephrology 6:1125-33, 1995; Ahn et al. Nature Genetics 3(4):283-91, 1993; and Cawthon et al. Genomics 9(3):446-60, 1991). Therefore, the structure of these elements is not conventional in the art and skilled in the art would therefore not recognize from the disclosure that applicant was in possession of the genus of nucleic acid, including genes, comprising SEQ ID NO: 1.

Since the disclosure fails to describe the common attributes or characteristics that identify members of the genus, and because the genus is highly variant, the disclosure of specific nucleotide sequences and the ability to screen, is insufficient to describe the genus. One of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe and enable the genus as broadly claimed.

Enablement

Claims 1-3 and 10-11 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for an isolated cDNA of a plant nitrilase *NIT1*, does not reasonably provide enablement for any and all nitrilase DNA (*NIT1*) of any species nor any isolated nucleic acid of less than 100 kb comprising a nucleotide sequence encoding a Nit1 protein. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make the invention commensurate in scope with these claims.

The claims are directed to any purified *NIT1* gene wherein said gene is a human gene or mammalian gene (Claims 1-3). The claims are further directed to any isolated nucleic acid of less than 100 kb comprising a nucleotide sequence encoding a Nit1 protein wherein said protein is a human Nit1 protein (claims 10-11).

The specification discloses that human and murine *NIT1* genes were cloned and characterized. Their exon-intron structure, their patterns of expression, and their alternative

mRNA processing were determined and the tissue specificity of expression of murine *FHIT* and *NIT1* genes were asserted to be nearly identical (page 2). The specification further discloses that the human and mouse *NIT1* genes are members of an uncharacterized mammalian gene family with homology to bacterial and plant nitrilases, enzymes which cleave nitriles and organic amides to the corresponding carboxylic acids plus ammonia (page 3). Figure 1 teaches a sequence comparison of human, murine, *D. melanogaster* and *C. elegans* Nit1 and Fhit1 proteins. Figure 6 sets forth a highly conserved sequence of human, murine, *D. melanogaster* and *C. elegans NIT1* gene (SEQ ID NO: 1). The specification further discloses that the mouse and human Nit1 amino acid sequences were 90% identical; the human Nit1 amino acid sequence was 58% similar and 50% identical to the *C. elegans* nitrilase domain and 63% similar and 53% identical to the *D. melanogaster* nitrilase domain (page 12 and Figure 1). However, the specification fails to teach or provide parameters, mechanistic characteristics, or classes of nitrilase domains for which one of skill in the art could reasonably predict that the *NIT1* gene encodes a functional Nit1 protein which exists in any species and which can be utilized to practice the claimed invention without undue experimentation due to the unpredictability of the function of various plant and bacterial nitrilases known in the art, as well as the absence of guidance provided by the specification as to any *NIT1* gene with functions in the same manner as the fusion gene, Fhit, of *D. melanogaster* and *C. elegans*.

Accordingly, in view of the quantity of experimentation necessary to determine the *NIT1* gene of any species or an isolated nucleic acid encoding any Nit1 protein, the lack of direction or

guidance provided by the specification as well as the absence of working examples with regards to the breadth of the claims directed to any *NIT1* gene or any isolated nucleic acid that is less than 100 kb comprising a nucleotide sequence encoding a Nit1 protein, it would have required undue experimentation for one skilled in the art to make the claimed invention as broadly claimed.

Claims 13-16 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. The factors to be considered have been summarized as the quantity of experimentation necessary, the amount of direction or guidance presented, the presence or absence of working examples, the nature of the invention, the state of the prior art, the relative skill of those in the art, the predictability or unpredictability of the art and the breadth of the claims. *Ex Parte Forman*, (230 USPQ 546 (Bd Pat. App. & Int. 1986)).

The claims are drawn to a method of treating or preventing a disease or disorder in a subject comprising administering to said subject a therapeutically effective amount of a molecule that inhibits or enhances Nit1 function (claims 13 and 14) interpreted as directed to gene therapy (See page 1). In further embodiment, the claims are drawn to a method of diagnosing or screening for the presence of or a predisposition for developing a disease or disorder in a subject comprising detecting one or more mutations in *NIT1* DNA, RNA or Nit1 protein derived from

the subject (claim 15). The claims are also drawn to a method of treating or preventing a disease or disorder in a subject by using a vector containing the *NIT1* gene coding sequence (claim 16).

The claims are not enabled as the specification does not provide guidance as to the dosage amounts, dosage frequencies, modes of delivery, vectors for delivery, appropriate expression levels and targeting to supply any type of therapeutic treatment. The specification discloses that a tumor suppressor gene *FHIT* encompasses the common human chromosomal fragile site at 3p14.2 and numerous cancer cell bi-allelic deletions (page 2). The specification further discloses that in human and mouse, the nitrilase homologs and Fhit are encoded by two different genes, *FHIT* and *NIT1*, localized on chromosomes 3 and 1 in human, and 14 and 1 in mouse, respectively (page 2). The specification discloses that neither the *in vivo* function of Fhit nor the mechanism of its tumor suppressor activity is known but that analysis suggest that the enzyme-substrate complex is the active form that signals for tumor suppression (page 3). The specification further supports the unpredictability of Fhit function by stating that although the frequent loss of Fhit expression in several common human cancers is well documented, and results supporting its tumor suppressor activity have been reported, the role of Fhit in normal and tumor cell biology and its mechanism of its action *in vivo* is unknown (page 14).

While the specification teaches the skilled artisan how to determine the enzymatic function of the Nit1 protein in the claimed compositions only on the basis of similar homology to the Fhit fusion gene of *D. melanogaster* and *C. elegans*, it fails to provide guidance to the skilled artisan on how to use the claimed methods for any treatment of any disease or disorder. In

particular, no protocol is described in the specification comprising administration of the polynucleotide or any other nucleic acid molecules that inhibit or enhance Nit1 function. The specification does not provide sufficient guidance as to the appropriate route of administration of any vectors for treating any diseases/disorders, the appropriate concentration of vectors, dosage, promoter to regulate expression for any treatment such that one of skill in the art could reproducibly, consistently, and effectively treat the patient in need thereof without undue experimentation.

Although the concept of gene therapy has potential, the realities of the parameters which will result in therapeutic benefit have not been achieved and are considered unpredictable. With regard to *in vivo* gene transfer, the specification provides no example or therapeutic methodology that would be encompassed within claims 13, 14 and 16. For example, Eck & Wilson (The Pharmacological Basis of Therapeutics, 1996) teach numerous factors complicate the gene delivery art which would not have been shown to be overcome by routine experimentation. These include, the fate of the DNA vector itself (volume of distribution, rate of clearance into the tissues, etc.), the *in vivo* consequences of altered gene expression and protein function, the fraction of vector taken up by the target cell population, the trafficking of the genetic material within cellular organelles, the rate of degradation of the DNA, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, and the protein's compartmentalization within the cell, or its secretory fate, once produced. These factors differ dramatically based on the vector used and the protein being produced, which cells

are target cells, and the disease and/or host being treated. It is further noted that Eck and Wilson support the importance of tailoring a gene therapy vector and method to specific diseases and/or disorders. See page 82, column 1, first paragraph. Furthermore, Eck & Wilson et al. review the state of the art for gene therapy for inherited disorders and discloses that "[t]he level of protein function necessary to achieve complementation of the defect varies widely among genetic diseases." See page 78, column 2, 2nd paragraph.

In addition, while progress has been made in recent years for gene transfer *in vivo*, vector targeting to desired tissues *in vivo* continues to be unpredictable and inefficient as supported by numerous teachings available in the art. For example, Miller (1995, FASEB J., Vol. 9, pages 190-199) review the types of vectors available for *in vivo* gene therapy, and conclude that "for the long-term success as well as the widespread applicability of human gene therapy, there will have to be advances...targeting strategies outlined in this review, which are currently only at the experimental level, will have to be translated into components of safe and highly efficient delivery systems" (page 198, column 1). Deonarain (1998, Expert Opin. Ther. Pat., Vol. 8, pages 53-69) indicate that one of the biggest problems hampering successful gene therapy is the "ability to target a gene to a significant population of cells and express it at adequate levels for a long enough period of time" (page 53, first paragraph). Deonarain reviews new techniques under experimentation in the art which show promise but states that such techniques are even less efficient than viral gene delivery (see page 65, first paragraph under Conclusion section). Verma (Sept. 1997, Nature, Vol. 389, pages 239-242) reviews vectors known in the art for use in gene

therapy and discusses problems associated with each type of vector. The teachings of Verma indicate a resolution to vector targeting has not been achieved in the art (see entire article). Verma also teaches appropriate regulatory elements may improve expression, but it is unpredictable what tissues such regulatory elements target (page 240, sentence bridging columns 2 and 3). Crystal (1995, Science, Vol. 270, page 404-410) also reviews various vectors known in the art and indicates that "among the design hurdles for all vectors are the need to increase the efficiency of gene transfer, to increase target specificity and to enable the transferred gene to be regulated" (page 409).

Specifically, the specification, on page 7, teaches only *in vitro* multiple tissue northern blots of *NIT1* cDNA probes. However, no further results are reported on the effectiveness of Nit1 protein function having any implication toward the treatment of any particular diseases or disorders. It is noted that, Orkin stress the importance of using relevant animal models for determining the effectiveness of therapeutic methodologies (p. 10 and 13). As such, the specification fails to provide any evidence which would provide a reasonable nexus to that of any particular diseases or disorders.

With regard to claim 15, directed to a method of diagnosing or screening for the presence of or a predisposition for developing a disease or disorder in a subject comprising detecting one or more mutations in *NIT1* DNA or RNA derived from the subject in which the presence of said one or more mutations indicates the presence of the disease or disorder or a predisposition for developing the disease or disorder, the specification fails to teach or suggest any methodology or

procedure for a method of diagnosing or screening of any mutations in *NIT1* DNA or RNA correlating to any disease or disorder in any subject as embraced by the claim. The specification only discloses that the pattern of *Nit1* expression was almost identical to the pattern of the expression of Fhit (Fig. 2A), supporting the hypothesis that the proteins may act in concert or participate in the same pathway (page 14, lines 1-4). However, the specification fails to discuss any methods of screening or diagnosing of any disease or disorder in any subject comprising detecting any mutations in *NIT1* DNA or RNA in which the presence of any said mutations would indicate the presence of any disease or disorder. The specification further fails to indicate that any mutations in *NIT1* DNA or RNA would even correlate to any disease or disorder. Thus, it would be unpredictable for one of skill in the art to identify mutations of *NIT1* DNA or RNA which would result in any disease or disorder as embraced by the claimed invention.

If claims 13-16 should be overcome by applicants arguments and/or evidence, claim 13-16 would be limited to use and specific therapeutic function of the exemplified Nit 1 nucleic acid sequence.

Accordingly, in view of the unpredictable and undeveloped state of the art, the lack of guidance or working examples which demonstrate or correlate to any therapeutic effect of the claimed methods, including the identification of any Nit1 mutations, and the breadth of the claims, the specification fails to teach any nucleic acid sequences which "enhance or inhibit" a Nit 1 protein as embraced by the claims directed to a method of treating or preventing any disease or disorder in any subject, including any are disease relevant Nit 1 mutations.

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1-3, 10-11 and 13-15 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 1, 10 and 11 are vague and indefinite for its recitation of "*NIT1* gene" because it is unclear from the specification as to what is encompassed within the claims as to "*NIT1*" gene since Figure 6 (SEQ ID NO: 1) is "NITD". Is *NIT1* the same as NITD? The metes and bounds of the claim cannot be determined. **Note** that claims 2-3 depends from claim 1.

Claims 13 and 14 are incomplete. While all of the technical details of method need not be recited, the claims should include enough information to clearly and accurately describe the invention and how it is practiced. The method of claims 13 and 14 are missing process steps. The method step needs to correlate to the preamble because it is unclear as to how inhibiting Nit1 function or enhancing Nit1 function would treat or prevent any disease or disorder.

Claim 15 is incomplete. While all of the technical details of method need not be recited, the claims should include enough information to clearly and accurately describe the invention and how it is practiced. The method of claim 15 is missing process steps. The method step needs to correlate to the preamble because it is unclear as to how mere detecting one or more mutations in *NIT1* DNA or RNA would diagnose or screen for the presence of or a predisposition for developing any disease or disorder.

Claim 16 is incomplete. While all of the technical details of method need not be recited, the claims should include enough information to clearly and accurately describe the invention and how it is practiced. The method of claim 16 is missing process steps. In addition, the method step needs to correlate to the preamble because it is unclear as to how using a vector containing *NIT1* gene coding sequence would treat or prevent any disease or disorder.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless --
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1 and 10 are rejected under 35 U.S.C. 102(b) as being anticipated by Hillebrand et al (May 8, 1996) Gene, Vol. 170 (2): 197-200.

The claims are drawn to any and all purified *NIT1* gene (claim 1) or any isolated nucleic acid of less than 100 kb comprising a nucleotide sequence encoding any Nit1 protein (claim 10).

Hillebrand et al disclose that a full-length genomic clone encoding the **complete** cluster of the *At* nitrilases 1-3 (NIT 1-3), including the respective promoter regions, has been isolated and sequenced. Thus, Hillebrand et al clearly anticipate claims 1 and 10 of the instant invention.

Conclusion

Claims 2-3, 11 and 13-16 appear to be free of the cited prior art of record because the cited prior art of record fails to teach or suggest a purified human or mammalian *NIT1* gene as

well as a method of treating, preventing, diagnosing or screening any disease or disorder using the same. However, these claims are subject to other rejections.

No claims are allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Gai (Jennifer) Mi Lee, whose telephone number is 703-306-5881. The examiner can normally be reached on Monday-Thursday from 8:30 to 5:00 (EST). The examiner can also be reached on alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Karen Hauda, can be reached on 703-305-6608. The FAX phone numbers for group 1600 are 703-308-4242 and 703-305-3014.

An inquiry of a general nature or relating to the status of the application should be directed to the group receptionist whose telephone number is 703-308-0196.

Gai (Jennifer) Lee
Patent Examiner
Art Unit 1600